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# *The Role of the Carotid Arteries in the Causation of Vascular Lesions of the Brain*

## *With Remarks on Certain Special Features of the Symptomatology*

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# THE ROLE OF THE CAROTID ARTERIES, IN THE CAUSATION OF VASCULAR LESIONS OF THE BRAIN, WITH REMARKS ON CERTAIN SPECIAL FEATURES OF THE SYMPTOMATOLOGY.[1]

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Introduction. In recent years, special attention has been given to the more exact localization of vascular lesions of the brain, and as a result a number of new syndromes have been added to the literature of this subject. The object of the present study is to emphasize the importance of obstructive lesions of the main arteries of the neck (the innominate, common carotid, and internal carotids), in the causation of softening of the brain, and more especially to urge the routine examination of these vessels in all cases presenting cerebral symptoms of vascular origin. In other words, the writer would advocate the same attitude of mind toward this group of cases as toward the intermittent claudication, gangrene, and other vascular symptoms of the extremities, and never omit a detailed examination of the main arterial stem.

Hemiplegia from occlusion of the intracranial branches of the internal carotid is one of the most frequent clinical pictures met with in medical practice, and yet apparently one of the rarest from obstruction of the main arterial pathway in the neck. However, when one considers that these vessels are frequently ignored both in clinical and pathological studies, it is not unlikely that many such conditions are overlooked.

The reason for our neglect in this respect is obvious, and springs from the assumption that the circle of Willis is sufficient to carry the blood into an obstructed vascular area when such obstruction is situated below the level of the communicating arteries. There is no question as to the correctness of this assumption in a large majority of cases, especially in young subjects with good hearts and elastic vessels. On the other hand, in those individuals with diseased and weakened hearts and sclerotic vessels, the necessary compensatory changes required for the establish-

[1] Presented at a meeting of the American Neurological Association, June, 1913.

ment of the collateral circulation may not develop, in which event any obstruction to the direct flow of blood through the carotid artery must be a considerable menace to the circulation of blood in the affected hemisphere. In such a case, even if the collateral circulation becomes established, it may prove insufficient for the needs of the hemisphere, because of the reduction in force and the consequent slowing of the blood current, when breaking down would follow in that portion of the brain commonly involved in failing cerebral circulation; the subcortical white matter, and the region of the basal ganglia. It is also obvious that even if actual softening does not occur, symptoms of diminished functional activity may follow with the characteristic picture of cerebral intermittent claudication.

We also know, that entirely apart from a weakened heart and arteriosclerotic vessels, obstruction of a carotid may produce permanent hemiplegia because of inadequate arteries of communication in the circle of Willis or other vascular anomalies.

It may be well to say, in this connection, that the focus of softening found at autopsy may be merely the breaking down at the distal portion of the arterial tree, where the circulation is weakest, exactly as in senile gangrene of the extremity; the important obstruction to the blood current being situated in the main trunk. Occasionally an area of softening in the region of the central ganglia is found with the vessels of the circle of Willis soft and patulous (Hunt[2]). In such cases the main artery of the neck may well come under suspicion, and should be carefully examined. The same is true of embolism, in which a central area of softening is found without demonstrable occlusion of the middle cerebral. In such cases it is usually assumed that the embolus has been broken up and undergone absorption. This explanation, however, is not always satisfactory, when the vessel is patulous and evidences of secondary thrombus formation are absent.

It would, therefore, seem proper in all cases with cerebral symptoms of vascular origin, to examine the pulsation of the carotids in the neck as possibly throwing some light on the source of the obstruction and rendering more exact our localization of the seat of the trouble in this group of cases. For this purpose a section of the carotid artery is readily accessible to palpation, extending from the lower border of the thyroid cartilage to the angle of the jaw; the main trunk divides into the external and internal carotid at the level of the hyoid bone, and the internal, the larger of the two vessels, is readily felt from this point to the angle of the jaw.

The writer has examined a number of cases during the past few months, at different ages, and suffering from a variety of diseases, and in all of them has found the force of the pulsation in these

[2] The Chronic Progressive Softening of the Brain, Amer. Jour. Med. Sci., June, 1906.

vessels to be practically equal on the two sides. The only exceptions noted were in a small series of hemiplegic cases of vascular origin, in which a definite and distinct *weakness of the carotid pulsation was noted on the side corresponding to the cerebral lesion.* These cases will be referred to later. It may be well to mention that rarely congenital inequalities in size may occur, and one carotid may even be absent as an extremely rare anomaly.

The symptomatology of occlusion of the common carotid artery is based largely on the study of those cases in which ligation was performed, an operation formerly much in vogue for a variety of indications. There are, comparatively speaking, only a few recorded cases of carotid obstruction from endarteritis, thrombosis, and embolism, and of these not a few were associated with aneurysmal dilatation of the arch of the aorta or its branches. We will review briefly these various groups of cases in order to show the different types of cerebral manifestations which may result when the main artery is occluded, and which, after all, is only a *resume* of clinical pictures with which we are perfectly familiar, and associate with the usual softening processes of the brain, but which are rarely considered as having a possible relation to the carotid artery in the neck.

After-effects of Ligation of the Common Carotid Artery. Ligation of the common carotid artery may produce merely temporary cerebral symptoms, such as unilateral headaches, vertigo, dilatation of the pupil, transient motor and sensory disturbances, which pass away as the collateral circulation becomes established. These are the so-called *immediate* symptoms, and in this group of cases, fortunately a large one, no permanent damage to the brain tissue results.

It would be interesting to know the subsequent cerebral histories of such patients, especially in the later period of life, when the degenerative changes in the circulatory system take place, and if the one hemisphere is more predisposed by reason of defective circulatory conditions to degenerative changes; but I am aware of no investigations bearing on this question.

The so-called *late* or permanent cerebral symptoms which come on after ligation, are the result of organic changes and vary in frequency in the statistics of different observers.

Pilz,[3] who collected and analyzed 520 cases in 1868, found that hemiplegia resulted in 50 cases.

LaFort's[4] statistics, published in 1875, were based on 370 cases, of which cerebral accidents of various kinds were noted in 100.

In Nieten's[5] series of 143 cases published in 1893, cerebral lesions occurred in 27 cases.

[3] Zur Ligatur der Arteria Carotis Communis. Arch. f. klin. Chir., 1868, Band ix, S. 257.
[4] L'Artere Carotid, Dict. Encyclop. des Sciences Méd. de Dechambre, 1875, T. xii.
[5] Inaugural Dissertation, Rome, 1893. (Cited by Lestolle.)

In Lestelle's[6] more recent study of 126 cases, cerebral lesions were noted in 11 per cent.

It must be remembered that all of the cerebral accidents cannot be attributed to the vascular occlusion, as other factors such as sepsis and meningitis are to be considered, but some idea of the uncertainty attending the establishment of the collateral circulation may be obtained and the dangers to the cerebral circulation from obstruction of the main arterial pathway.

The late manifestations consist usually of hemiplegia with or without hemianesthesia, aphasia, convulsions, and the other well-known symptoms resulting from obstruction in the distribution of the middle cerebral artery. Occasionally, blindness or even destruction of the eye may follow on the side of the ligation, from impairment of the circulation in the ophthalmic artery. This is an interesting symptom, and will be referred to later as a diagnostic sign of possible importance.

Before leaving the subject, mention must be made of those cases in which ligation of the carotid has been performed on both sides without ill effects, providing sufficient time has elapsed for the proper establishment of the first collateral circulation, which then conducts the blood to the brain after the second ligation. These cases show what an extraordinary power of adjustment is sometimes possible in the cerebral circulation. It is, however, by no means a certain mechanism, and in a given case one can never foretell what may be the effect of the ligation on the cerebral structures.

Thrombosis of the Carotid Artery from Injury. The carotid artery may become thrombosed after injuries to the neck, such as stab wounds, gunshot wounds, and the like.

The following cases have come under my personal observation, and are reported for their symptomatological interest:

Case I.—A man, aged thirty-two years, was brought to the Hudson Street Hospital on September 31, 1903, immediately after receiving a stab wound on the left side of the neck. There was a small puncture wound in the middle of the left side of the neck, over the sternocleidomastoid muscle, from which there was practically no bleeding. At the site of the wound there was a soft swelling about the size of a pigeon's egg, which was pulseless and yielded no bruit. The patient was conscious and able to tell the history of the accident. Immediately on being stabbed he fell to the ground with vertigo, faintness, and numb feeling on the right side. On admission to the hospital, immediately after the accident, symptoms of right hemiplegia were already apparent.

On October 5, I made an examination at the request of Dr. Stimson. The man was somnolent and apathetic. His pupils were

[6] Des Accidents Cerebraux consecutif a la ligature de l'artere Carotid Primitive. Thése de Paris, 1903.

equal and reacted to light. The right side was completely hemiplegic, with considerable loss of sensibility extending to the middle line. The tendon reflexes were present. The abdominal and cremasterie reflexes were abolished on the right side. Babinski phenomenon were present on the right side. There was moderate conjugate deviation of the head toward the left side. The left temporal artery was pulseless. On the right side distinct pulsations were palpable.

Ophthalmoscopic examination shows the left papilla to be pale, the veins moderately full, and the arteries distinctly smaller and less prominent than on the right. The heart sounds were good; pulse, 72; temperature, 101°. The diagnosis of thrombosis of the common carotid artery was made.

October 6. Operation. An incision was made along the left sternocleidomastoid muscle. After a small blood-clot had been removed, the jugular vein was found punctured and was ligated. The common carotid artery was almost completely divided just below the bifurcation and was the seat of a firm thrombosis. The artery was ligated above and below the injury and the severed portion removed.

In the course of the next few days, the patient became gradually more stuporous, and terminal pneumonia developed, with rising temperature, death occurring thirteen days after the injury. The brain could not be examined.

Case II.—A boy, aged fourteen years, was brought to the New York Hospital, immediately after an injury, on April 30, 1909. He was in an unconscious condition, paralyzed on the right side, and bleeding profusely from a punctured wound on the lower lip and floor of the mouth. He had been injured by falling from a fence upon the iron tip of an umbrella, which entered the left side of the lower lip, passing through the floor of the mouth. According to bystanders he was able to rise, but almost immediately fell to the ground, unconscious, where he remained until picked up by an ambulance surgeon a few minutes later.

Examination, at the request of Dr. Hartley, May 1, 1909, 2 P.M. In addition to the punctured wound of the lip and floor of the mouth there is a swelling beneath the left sternomastoid muscle, just below the angle of the jaw, which does not pulsate. He is unconscious, but can be aroused for brief periods, and is then irritable and somewhat resistant to examination. There is a complete right hemiplegia with diminution of the pain sense on the right side. The right knee-jerk is absent, the left present; both Achilles-jerks and the arm reflexes are present. Babinski present on the right side. Abdominal and cremaster reflexes present on both sides. Pupils are equal and react to light. Heart is normal; systolic blood-pressure is 115 mm. He vomited several times during the day. Temperature, 101°; pulse, 116. Lumbar

puncture was performed and three drams of clear fluid remained, which was entirely negative.

May 2. The patient is unconscious, and cannot be aroused. The swelling on the left side of the neck is soft and baggy, and has increased slightly. It does not pulsate. There is neither rigidity of the neck nor conjugate deviation of the head or eye. Pupils are still equal and react to light. There is no pulsation in the left temporal artery. Paralysis and reflexes are as noted in first examination.

*Ophthalmoscopic Examination.* The arteries of the left disk are smaller than those of the right, and do not pulsate. The veins are somewhat fuller on the left side. The left papilla is distinctly paler than the right. Death occurred May 2, at 10.30 P. M.

*Autopsy Note.* The carotid artery of the neck was dissected out and found to be punctured at the bifurcation, and completely thrombosed. The examination of the brain showed some swelling of the convolutions over the parietal and temporal region, and the subjacent white matter was softened and broken down. On opening the corpus collosum, the floor of the left lateral ventricle was found irregularly mottled and soft. The circle of Willis was free except for thrombosis of the middle cerebral artery. Both the anterior and posterior communicating arteries were present and patulous.

*Remarks:* These cases are similar in etiology and symptomatology, and are of interest in the present discussion, as showing the development of immediate cerebral symptoms after puncture wounds of the carotid artery. No doubt this was due to the sudden interference with the cerebral circulation on the wounded side. It is hardly possible that embolism could have occurred so promptly. The steady advance of the softening process terminating in death, was probably due to a slow extension of the thrombus into the cerebral vessels, and which could be demonstrated in Case II. It is to be noted that in both cases there was an absence of pulsation in the temporal artery on the affected side, showing that the external carotid artery was occluded. I would particularly emphasize the slight and distinct vascular changes in the optic disk on the side of the injury present in both of the cases. Such milder disturbances of the circulation in the optic disk as well as optic atrophy, which sometimes develops, may serve to throw light on the localization of the obstruction, showing the lesion to be situated below the ophthalmic branch of the internal carotid.

Primary Thrombosis of the Carotid Artery (Arteriosclerosis, Endarteritis). Spontaneous thrombosis of the carotid from disease of the vessel wall occurs but rarely, and there are but few cases recorded in the literature of this subject. Among the earlier

writers, cases were described by Chevers,[7] Gull,[8] Todd,[9] Savory,[10] and Crisp,[11] usually as a complication of aneurysm of the arch of the aorta or its larger branches. A more recent case of this nature has been recorded by W. Erb, Jr.[12]

Kussmaul[13] has also described two cases of spontaneous and gradual occlusion of the carotid. One of endarteritis, with complete thrombosis of the vessel, in which the only symptom noted was headache, and a few days before death, visual disturbances. In the second case the left common carotid was occluded and the external carotid was pulseless. The symptoms produced were left-sided headache, vertigo, and epileptiform attacks. The left optic disk was pale, the veins full, and the arteries small. There was no paralysis. He notes that the communicating arteries were but poorly developed.

In Penzoldt's[14] case of thrombosis of the right carotid artery the initial symptom was sudden blindness, the sight of the right eye remaining permanently defective and the disk atrophic. Later a left hemiplegia developed. Autopsy showed complete thrombosis of the right common carotid, with a large area of softening in the right hemisphere. The external carotid was free from thrombosis formation.

Another case of optic atrophy, with contralateral hemiplegia from occlusion of the carotid, is recorded by Guthrie and Mayou.[15] The left carotid was pulseless and the obstruction may have been due to embolism, as the heart action was irregular and a systolic bruit was present.

It is important to remember that symptoms of chronic progressive hemiplegia may follow thrombosis of the carotid artery. Cases of this nature have been described by Oppenheim,[16] Trenel[17] and Brissaud.[18]

In Brissaud's case, a man aged forty-seven, developed gradually the symptoms of hemiplegia, and died in coma. The autopsy revealed a large area of white softening occupying the inferior two-thirds of the Rolandic area, which had a special edematous character. The internal carotid was the seat of an annular endar-

[7] Effects upon the Cerebral Circulation of Obliteration of one Common Carotid Artery, London Gaz., i, new series, p. 146.

[8] Case of Occlusion of the Innominate and Left Carotid, Guy's Hosp. Rep., 1855, i, 12.

[9] Med.-Chir. Trans., xxvii, 301.

[10] Ibid., 1856.

[11] Cited by Pilz, Arch. f. klin. Chir., Band ix, S. 403.

[12] Ein Fall von Ausgedehnter Gehirn erweichung bei totaler Obliteration der Carotis Communis Sinistra, Münch. med. Woch., 1904, li, 947.

[13] Zwei Fälle von spontaner Allmaliger Verschleisung grosser Hals arterien Stamme, Deutsch. Klinik, 1872, No. 51.

[14] Ueber Thromboso der Carotis, Deutsch. Arch. f. klin. Med., 1881, xxviii, 81.

[15] Right Hemiplegia and Atrophy of the Left Optic Nerve, Proc. Royal Soc. Med., 1907–1908, i, 180.

[16] Lehrbuch der Nerven Krankheiten, 1908, ii, 942.

[17] Cited by Oppenheim.

[18] L'Hemiplegie Progressive, Rev. Neur., 1898.

teritis, which had diminished, but not entirely occluded the caliber of the vessel.

Embolism of the Carotid Artery. Embolic occlusion of the carotid in the neck appears to be even rarer than thrombosis. The only recorded cases are those of Cohn,[19] Eichhorst,[20] and Haffner.[21]

In Haffner's case there had occurred an embolic occlusion of the left brachial artery in October, 1892. In 1895 there appeared suddenly parietal headache, vertigo, and visual disturbances; no paralysis. A painful swelling developed just below the left ear, and the left carotid and temporal artery were pulseless. Ophthalmoscopic examination was negative. Two years later, in March, 1897, there developed gradually, hemiplegia of the right side, which became complete in about a week. This was accompanied by some sensory loss as well. The patient became apathetic and later demented. Autopsy showed an organized thrombosis of the left carotid, which extended a little beyond the bifurcation. There were small foci of softening in the centrum semiovale on the left side.

Hemiplegia Associated with Diminished Pulsation of the Carotid Artery in the Neck. Under this heading I would call attention to the occurrence of diminished pulsation of the carotid artery in the neck on the side of the softening in cases presenting the symptoms of thrombotic hemiplegia.

Among a series of twenty cases of hemiplegia occurring in advanced life, which I examined for this symptom in the neurological service of the Montefiore Home for Chronic Invalids, it was possible to demonstrate its presence in four cases, all of which presented the clinical picture of an extensive lesion of the hemisphere: hemiplegia with contractures, hemi-sensory disturbances and mental deterioration. The optic nerves were normal on both sides in all four cases.

While inequality of pulsation on the two sides might be accidental, its occurrence in four cases all presenting the symptoms of extensive brain softening is rather significant, and the thought naturally arises that some obstructive lesion of the vessel or its entrance in the arch of the aorta has interfered with the free flow of blood to the brain, which in old subjects with weakened heart would be a predisposing factor in the production of senile softening of the brain.

Of course, further observations and pathological studies will be necessary in order to determine with certainty this point. If, however, these observations are confined, such a diminution

[19] Klinik der Embolischen Gefäss Krankheiten, 1860, S. 364.

[20] Ueber Emboli der Carotis Communis, Med. Klinik., 1907, iii, 885.

[21] Obliteration der Carotis Communis Sinistra in folge von Embolischer Arteritis bei Herzfehler, Deut. Arch. f. klin. Med., lx, 523.

in the pulsation of one of the carotids, would be a symptom of some prognostic value before the development of hemiplegia.

Some Concluding Remarks. I would urge that all cases presenting cerebral symptoms of vascular origin, that the main arteries of the neck be carefully examined for a possible diminution or absence of pulsation. Obstructive lesions of these vessels are apparently rare, but it seems certain that cases are overlooked from failure to make clinical and pathological examinations from this point of view.

Probably in the large majority of cases, a sudden obstruction may occur without softening, producing nothing more than mild, and transient cerebral symptoms. On the other hand, this may so cripple the cerebral circulation in advanced life, that functional deficiencies result and the one hemisphere may be more predisposed to softening processes than the other.

I would emphasize the occurrence of *diminished pulsation of the carotid on the side of the softening as an occasional symptom in cases of thrombotic hemiplegia*, and suggest that the impairment of circulation through the carotid so interferes with the general circulation of the hemisphere as to predispose to thrombus formation and encephalomalacia. A diminished pulsation from this point of view would merit a place as a prognostic factor of importance.

There are various clinical pictures resulting from obstruction of the carotid, similar in symptomatology to those accompanying the intracranial vascular degeneration and diseases. Among these are hemiplegia, chronic progressive hemiplegia, the chronic progressive softening of the brain (Wernicke), and optic atrophy with contralateral hemiplegia.

The distinguishing feature is an absence of the carotid pulse on the side corresponding to the lesion. Should the thrombosis extend to the bifurcation and include the external carotid, the temporal artery on the affected side will be pulseless.

I would also particularly emphasize the occurrence of *unilateral vascular changes, pallor, or atrophy of the disk with contralateral hemiplegia* in obstruction of the carotid artery.[22]

It is also important to note, that the visual disturbances and vascular changes in the optic nerve may precede other organic cerebral symptoms, the collateral circulation failing to develop in the distribution of the ophthalmic artery.

Unilateral headaches and vertigo, especially in assuming the

[22] This syndrome has also been described with intracranial vascular disease: Gowers (Embolism of the Middle Cerebral and of the Central Artery of the Retina, with Autopsy, Lancet, 1875, p. 794); Cadwallader (Unilateral Optic Atrophy and Contralateral Hemiplegia Consequent on Occlusion of the Cerebral Vessels, Jour. Amer. Med. Assoc., 1912, p. 2248); Guthrie and Batten (Unilateral Atrophy of the Optic Nerve Associated with Hemiplegia of the Opposite Side, Trans. Chir. Soc., xxxvi, 52); Elschung (Ueber die Embolie der Arteria Centralis Retina, Arch. f. Augenheil., 1891, xxiv, 65); and Starr (Diseases of the Nervous System, 1910, p. 500).

upright posture, epileptiform attacks, failing memory, attacks of threatened hemiplegia, cerebral intermittent claudication, are some of the vascular symptoms which should suggest the possibility of carotid obstruction.

In the realm of pathology, such conditions as chronic edema, indurative, atrophic, and softening processes of the brain, in which the main vessels of the circle of Willis are free from obstruction, should always give rise to the suspicion of impaired circulation in the carotid artery.

These various symptoms and syndromes and their underlying pathological conditions are all perfectly well-known, but as was remarked in the introduction to this paper, we instinctively associate them with intracranial vascular disease.

It may add to our knowledge of encephalomalacia and the accuracy of vascular localization if the main vessel in the neck is also considered.